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EXAMINER	
SALMON, KATHERINE D	
ART UNIT	**PAPER NUMBER**
1634	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	03/13/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Office Action Summary	Application No.	Applicant(s)	
	10/659,519	SIDRANSKY ET AL.	
	Examiner	Art Unit	
	Katherine Salmon	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *06 December 2006*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *12-24* is/are pending in the application.

4a) Of the above claim(s) *20-24* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *12-19* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. This action is in response to the papers filed 12/06/2006. Claims 12-24 are pending. Claims 20-24 are withdrawn as being directed to nonelected inventions.

2. The following rejections are necessitated by amendment. Response to arguments follows.

3. A complete reply to the final rejection must include cancellation of nonelected claims and subject matter or other appropriated action (37 CFR 1.144) See MPEP §821.01.

4. This action is FINAL.

Withdrawn Rejections

5. The rejection of the claims under 35 USC 112, second paragraph made in section 4 of the previous office action is moot in view of the amendments to the claims.

6. The rejection of the claims under 35 USC 102(a) as being anticipated by Herbert et al. in section 6 of the previous office action is moot in view of the amendments to the claims.

7. The rejection of the claims under 35 USC 102(b) as being anticipated by Tulchinsky et al. in section 7 of the previous office action is moot in view of the amendments to the claims.

New Rejections Necessitated by Amendment

Claim Objections

8. Claim 13 is objected to because of the following informalities: Truncation is spelled "truncation" in the claim. Appropriate correction is required.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

9. Claims 12-19 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 12-19 are indefinite over the phrase "the 5' ALT promoter of the p16 gene". It is not clear as to what constitutes this promoter, because the specification and art do not teach the "5' ALT promoter of the p16 gene". The instant specification discloses the 5' ALT gene.

Claims 13 and 16-17 are unclear. Claim 13 recites the limitation "the second product" in line 3. There is insufficient antecedent basis for this limitation in the claim. The claim is drawn to step a of Claim 12 which does not contain the phrase "second product". It is suggested that the claim be amended to provide proper antecedent basis. Further, it is unclear if "second product" is referring to "second amplification product" found in step c of Claim 12 or another product.

Claims 13 and 16-17 are unclear. Claim 13 recites the limitation "the p16 gene product" in line 4. It is unclear which product is consider "the p16 gene product". Claim 12 recites amplification of an exon 1 region, exon 2 regions it is unclear if "the p16 gene

product" is the exon 2 regions in the absence of exon 1, the exon 1 region, the exon 2 regions, or the exon 1 and 2 region.

Claim Rejections - 35 USC § 112-New Matter

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

10. Claims 12-19 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The cited passages have been reviewed but do not appear to provide support for the "5' ALT promoter region of the p16 gene", "truncated p16 gene product", the presence of hypermethylation of the 5" ALT promoter of the p16 gene is associated with the presence of any truncated p16 product, and addition of the demethylation agent in step a.

The amended Claims 12-19, reference the 5' ALT promoter region of the p16 gene (Claim 12 step b and c). Upon review of the newly added recitation, the

specification does not appear support the 5' Alt promoter region of the p16 gene. The instant specification describes a 5' ALT gene which is 30 kbs upstream of exon 1 of the p16 gene.

The amended Claims 12-19 reference a "truncated p16 gene product" in Claim 12 step C. However the specification does not appear to support a "truncated p16 gene product". The specification asserts an alternative splice variant of the p16 gene in which a novel 5' sequence was spliced precisely onto the first base of exon 2 of the p16 (p. 15 paragraph 155). The specification asserts that this 268 bp fragments (5'ALT) was not in frame with the coding sequence of exon 2 and 3 of p16 and the original exon 1 was excluded from this alternative transcript but does not indicate that this is a truncated p16 gene product (p. 15 paragraph 155).

The amended Claims 12-19 reference the presence of hypermethylation of the 5' ALT promoter of the p16 gene is associated with the presence of any truncated p16 product. The instant specification does not teach an association with any truncated product.

The amended Claims 13 and 16-17 reference contacting the sample with a demethylating agent in the method step of primer extension of the sample's exon 1 and 2 regions wherein the second product is detectable when methylation of the promoter results in truncation of the p16 gene product. The reply points to paragraph 193 of the instant specification. This paragraph indicated that adding a demethylation agent to a sample prior to detection of mRNA that the p16 mRNA is detectable (paragraph 193).

The specification does not show contacting a sample with a demethylating agent in the same step as A.

These amendments to the claims, therefore, constitute new matter.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

11. Claim 12-19 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for

12. A method comprising:

a) contacting a tumor sample comprising RNA with the oligonucleotide primer consisting of SEQ ID No. 15 which amplifies the exon 1 region of p16 gene and the primer consisting of SEQ ID No. 16 which amplifies the exon 2 region of the p16 gene under conditions for primer extension and producing an amplified product

b) contacting the amplified product with the primer consisting of SEQ ID No. 8 which binds to and extends the 5' ALT gene

c) detecting the presence of a 428 bp amplification production containing the amplified portions of exon 1 and 2 or detecting the presence or detecting the amplification product of only exon 2 when the 5'ALT gene is present.

13. The method of claim 12 wherein before the tumor sample is extended and amplified, a demethylation agent is added to the tumor sample and wherein in step c, there is detection of exon 1 and a 428 bp fragment is produced and wherein when the demethylation agent is not added to the sample there is no detection of exon 1.

, does not reasonably provide enablement for a method of detecting methylation of a p16 gene by extension of any fragment of exon 1 and 2 wherein the absence of exon 1 is indicative of hypermethylation of 5'ALT promoter of the p16 and the presence of a truncated product. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make or use the invention commensurate in scope with these claims.

Factors to be considered in determining whether a disclosure meets the enablement requirement of 35 USC 112, first paragraph, have been described by the court in *In re Wands*, 8 USPQ2d 1400 (CA FC 1988). *Wands* states at page 1404,

> "Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in Ex parte Forman. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims."

The nature of the invention and breadth of claims

Claim 1 is drawn to a method of detecting methylation of a p16 gene by amplification of the exon 1 and exon2 region wherein the absence of the exon 1 region is indicative of a truncated product. Claim 13 is drawn to contacting the sample with a demethylating agent wherein the second product (exon 1) is detected when methylation of the promoter results in truncation of the p16 gene product. Claims 14-15 define the sample. Claim 16 is drawn to a method wherein methylation of the p16 gene is indicative of a neoplasm. Claim 17 defines the neoplasm. Claim 18 define the sample. Claim 19 defines the amplification reaction.

The claims encompass primers which extend and amplify any fragment of exon 1 and 2. The specification indicates that specific primers are used to amplify a 428 bp region of the p16 gene when the p16 gene is not methylated (p. 13 paragraph 127-131). It is unpredictable that any fragment of the p16 exon 1 and 2 is correlated to methylation.

The specification asserts that when the 5' ALT gene is spliced into the region right before exon 2 then the product of exon is not amplified (paragraphs 135-136). Therefore the specification indicates that the specific amplification of the entire exon 1 and 2 region is affected by methylation because the 5' ALT gene splices into the sequence and therefore the two specific primers used can not amplify the exon 1 region and the exon 2 region. The claims, however, are broadly drawn to amplification of any exon 1 or 2 region, it is unpredictable that methylation and the insertion of the 5' ALT gene into the p16 gene would affect the amplification of any region of the exon 1 or 2 by any primer.

The claims are drawn to extending sequences from the 5' ALT promoter region of the p16 gene, however, the specification is drawn to the detection of the 5' ALT gene that is spliced into the p16 gene. It is unclear if the 5' ALT promoter region of the claims is the same as the 5'ALT gene. The specification does not describe the 5'ALT genes as the promoter region.

The claims are broadly drawn to further contacting the sample with a demethylating agent. The claims are drawn to detecting the second product in the presence of the demethylating agent when methylation of the promoter results in

truncation of the p16 gene product. As written the claims add the demethylation agent after extension of exon 1 and 2. It is unclear that the demethylation agent would have any effect on already amplified fragments of nucleic acids.

The specification discloses adding demethylating agent for three days to a sample and then detecting p16 mRNA (paragraph 193). Wherein the p16 mRNA was detected because there was no methylation. The claims, however, are drawn to contacting the sample with a demethylating agent in the same step of amplifying nucleic acid regions, which means that the agent can be added after extension. Further, the claims are drawn to detection of exon 1 (the second product) when methylation of the promoter results in truncation. The prior claim and the specification indicate that exon 1 is not detected when the p16 gene is methylated, however, Claim 13 indicates that exon 1 is present when the p16 gene is methylated.

Further, it is unclear the degree of methylation which must be in the sample in order to detect methylation by absence of exon 1. The specification discloses that in partially methylated cancer cell lines exon 1 and 2 were expressed (paragraph 193). Therefore the specification asserts that in the presence of exon 1 there is methylation of the p16 gene to some degree. Therefore the claims are drawn to any methylation whereas the specification indicates that the absence exon 1 is due to aberrant methylation.

The claims are broadly drawn to a method wherein the sample is contacted with the dimethylation agent and the second amplification product is detected then the methylation of the p16 gene is indicative of any neoplasm. However, the specification

does not show any p-values for this association. In Table 2, cell lines from cancers have intact p16 (assuming that intact p16 has both the full exon1 and exon 2) (p. 18). As discussed below, the art teaches that detection of methylation can be associated with aging, therefore, it is unclear if detection of any methylation is indicative of neoplasm. It is therefore unpredictable that any methylation detection is indicative of any neoplasm because the table indicates that methylation is not detected in all neoplastic tissue samples.

Nature of the Invention

The claims encompass method for detecting any fragment of exon 1 and 2 and the association of the lack of exon1 with any neoplasm. The invention is in a class of invention, which the CAFC has characterized as "the unpredictable arts such as chemistry and biology." Mycogen Plant Sci., Inc. v. Monsanto Co., 243 F.3d 1316, 1330 (Fed. Cir. 2001).

Guidance in the Specification

The specification does not provide any specific guidance as to how to predictably detect methylation in a sample population. The tables provided by the specification and the guidance in the specification indicate that in some cases methylation can not be detected by observing an absence of the entire exon 1 region in the p16 gene.

The specification asserts the prior art has shown abnormalities of p16 gene in primary tumors of certain cancers (p. 3 lines 3-5). The specification asserts in eukaryotic cells methylation of cytosine residues immediately 5' to a guanosine occurs

predominantly in CG poor regions (p. 3 lines 18-19). The specification asserts discrete regions of CG dinucleotides (CpG islands) are unmethylated in normal cells and methylation of the 5' regulatory regions lead to transcriptional repression (p. 3 lines 22-23).

The specification asserts methylated cell lines express an abundant, shortened p16 transcript devoid of exon 1 coding sequence (p. 9 last paragraph). The specification asserts hypermethylation of the 5'CpG island of p16 is frequent in cell lines and primary tumors of common human neoplasms (p. 10 1st paragraph). The specification asserts DNA methylation can occur in neoplasms with homozygous deletion (breast, renal) and those not associated with loss of p16 (colon and prostate) (p. 10 1st paragraph). The specification asserts hypermethylation in the p16 promoter region is a common abnormality of p16 in human cancers (p. 10 1st paragraph).

Working Examples

The examples provided by the specification, fail to provide guidance as to the detection of methylation using amplification of exon 1 and 2. The examples indicate that in some samples there is a lost of exon 2 associated with methylation. Further, the examples do not provide a clear guidance to determine if the detection of any loss of exon 1 is associated with any neoplasm.

Example 1 part 1: The specification asserts cells from head and neck cancer cell lines, lung cancer cell lines, pancreatic adenocarcinomas cell lines were extracted (p. 47 last paragraph).

Example 1 part 2 and 3: The specification asserts fragments of exon 1, 2, and 3 were amplified (p. 48 and 49).

Example 6: Table 1 presents 5' CpG island methylation related to allelic status and sequence analysis of the p16 in the cell lines. The p16 sequence indicates the majority of the primary human cancers have the wild-type p16 sequence (p. 61). This indicates p16 with exon 1 present (wildtype) would be observed in primary human cancers, therefore it is unpredictable to make an association of a mutant p16 gene (absent of exon 1) with cancers.

Example 7: The specification asserts sequence analysis of exon 1 and 2 of p16 in cell lines showed only one mutation in a HNSCC cell line (p. 62 2nd paragraph). The specification asserts the mutation caused exclusion of exon 2 but the line contained an unmethylated 5' CpG island since methylation and point mutation are independent modes of gene inactivation (p. 62 2nd paragraph). As such it is unpredictable that neoplasm or methylation can be associated with absence of exon 1. The example provided by the specification indicates that same methylated samples do not amplify exon2 of the p16 gene.

Example 10: The specification asserts detecting de novo methylation of p16 in tumor cells (p. 65 last paragraph). The specification asserts 4 NSCLC show de novo methylation whereas one does not exhibit methylation (p. 66 Figure 6b). The specification asserts 7 of 25 NSCLC showed aberrant methylation of p16 whereas 21 control samples did not show detectable methylation (no p value provided) (p. 66 last paragraph). This is unpredictable because the specification fails to show a significant

association of the methylation site of p16 and any tumor cell. It is unclear if 7 or 25 tumor cells would be statistically significant correlation of neoplasm to detection of methylation.

Example 11: The specification asserts Exon 1 of p16 lies in a CpG island which is unmethylated in normal tissue (p. 67 1st full paragraph). Table 2 shows inactivation of p16 in cell lines and primary tumors (p. 69). The specification asserts a correlation of methylation to cancer. There is no p value for the number of cell lines or tumors which had a methylated p16 region so it is unclear if there is a correlation. For example the 6 colon adenoma tumors tested only 1 was methylated. Therefore, it is unpredictable to correlate any tumor with methylation. Further, the specification asserts some primary colon cancers had hypermethylated p16 alleles while others had unmethylated alleles (p. 70 1st paragraph last sentence). It is unpredictable to detect ANY neoplasm by detection of methylation. The specification shows some tumor cell lines association with increased methylation and other tumors (colon adenoma) where the association is not clear.

The unpredictability of the art and the state of the prior art

The current art teaches that methylation is not only caused by neoplasms, but that methylation can be detected in normal tissue. This indicates that detection of methylation does not indicate neoplasic tissue. The current art teaches detection of methylation is indicative of not only neoplasm but also aging of normal cells. Yates et al. (Oncogene 2006 Vol 25 p. 1984) teaches that methylation increases with age and

malignancy (abstract). Yates et al. teaches that methylation was detected in urine DNA from patients with and without bladder cancer (Abstract). Yates et al. teaches aberrant methylation is not cancer specific and can be found in a normal ageing cell population (p. 1985 1st column 1st paragraph). Yates et al. teaches the overall knowledge of the molecular mechanisms of DNA methylation in health and cancer remains poor and one uncertainty is the extent of aberrant DNA methylation in nonmalignant tissue and the association between ageing and aberrant DNA methylation (p. 1985 last paragraph).

Quantity of Experimentation

The quantity of experimentation in this area would be extremely large since there is significant number of parameters that would have to be studied. To practice the invention as broadly as it is claimed, the skilled artisan would have to determine which fragments of exon 1 are amplified when there is not methylation versus the fragments are absence when the tissue is methylated. Further the skilled artisan would have to determine a clear correlation between the presence of exon 1 and 2 and methylation. The specification indicates that in some instances exon 2 is absent in methylated tissue, whereas the claims are drawn to the absence of exon 1. The skilled artisan would have to determine the correlative association of every possible neoplasm and detection of methylation.

The skilled artisan would need to perform undue experimentation to determine which parts of the amplified p16 fragments are affected by methylation. There seems to be no clear pattern in the specification to guide the artisan to be able to correlate the absence of any part of exon 1 with methylation. The skilled artisan would need to perform undue experimentation to determine if detection of methylation by amplification

of p16 gene is indicative of ANY neoplasm when the art shows that methylation is also indicative of other cellular processes such as aging.

To use the invention as presented would require a large amount of inventive effort, with each of the many intervening steps, upon effective reduction to practice, not providing any guarantee of success in the succeeding steps.

Level of Skill in the Art

The level of skill in the art is deemed to be high.

Conclusion

Case law has established that '(t)o be enabling, the specification of a patent must teach those skilled in the art how to make and use the full scope of the claimed invention without 'undue experimentation.'" *In re Wright* 990 F.2d 1557, 1561. *In re Fisher,* 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970) it was determined that '(t)he scope of the claims must bear a reasonable correlation to the scope of enablement provided by the specification to persons of ordinary skill in the art". The amount of guidance needed to enable the invention is related to the amount of knowledge in the art as well as the predictability in the art. Furthermore, the Court in *Genetech Inc. v Novo Nordisk* 42 USPQ2d 1001 held that '(I)t is the specification, not the knowledge of one skilled in the art that must supply the novel aspects of the invention in order to constitute adequate enablement".

Thus the applicants have not provided sufficient guidance to enable a skilled artisan to make the claimed invention in a manner reasonably correlated with the claimed method of detecting methylation using any amplified fragment of exon 1 and 2 wherein the absence of the fragments of exon 1 is indicative of methylation. Further the specification does not provide guidance to the correlation of the detection of methylation

to ANY neoplasm. The skilled artisan would have to perform undue experimentation to determine the relationship of the presence or absence of any fragment of exon 1 or 2 and methylation. The skilled artisan would have to perform undue experimentation to determine the relationship of ANY neoplasm and detection of methylation. Thus given the broad claims in an art whose nature is identified as unpredictable, the unpredictability of that art, the large quantity of research required to define these unpredictable variables, the negative teachings in the art, and the lack of guidance provided in the specification balanced only against the high skill level in the art, it is the position of the examiner that it would require undue experimentation for one of skill in the art to perform the method of the claim as broadly written.

Response to Arguments

The reply traverses the rejection. The reply asserts that neoplastic cells in which truncated p16 gene product can be detected can produce a full length p16 gene after demethylation (p. 6 3rd paragraph). The reply asserts that this reversal of truncation is associated with neoplastic cells and can be detected by the method in any neoplasm (p. 6 3rd paragraph). The reply asserts that this association was observed in many types of neoplastic tissue and it correlates with 5' ALT promoter methylation via detection of differential production of full p16 gene products (p. 6 last paragraph).

These arguments have been fully considered but have not been found persuasive.

The claims are not drawn to detection of a full length p16 gene after demethylation and detection of a truncated gene when methylated. Further, it is unclear if the "reversal" of expression of the p16 gene is indicative of neoplasm or indicative of aberrant methylation which may be caused a many factors including neoplasm and aging. There is no example in the specification to show that this reversal of the p16

product occurs only in neoplastic cells. It is further unclear if any neoplasm is indicative of methylation because the specification shows that some neoplasms show a normal expression of the p16 gene and some have partial methylation but no change in expression of the p16 gene.

Conclusion

12. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

13. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Katherine Salmon whose telephone number is (571) 272-3316. The examiner can normally be reached on Monday-Friday 8AM-430PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on (571) 272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Katherine Salmon
Examiner
Art Unit 1634

CARLA J. MYERS
PRIMARY EXAMINER